Orthogonal electrocardiogram, apex cardiogram, and atrial sound in normotensive and hypertensive 50-year-old men

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The prevalence of signs of heart involvement was studied non-invasively in a group of untreated hypertensives (n=35) and a reference group (n=73), all derived from a random population sample of 50-year-old men. Signs of left ventricular hypertrophy were studied by means of orthogonal electrocardiography and conventional electrocardiography. Signs of decreased distensibility of the left ventricle were studied by apex cardiography and registration of atrial sounds.

Left ventricular hypertrophy among hypertensives was significantly more common according to orthogonal electrocardiography (33%) than according to conventional electrocardiography (9%), indicating that the former may be a better method for detection of left ventricular hypertrophy than the latter. In the hypertension group the amplitude of the R wave in lead X on orthogonal electrocardiography was positively correlated to casual diastolic blood pressure (r=0·40) and to diastolic blood pressure after one hour's rest (r=0·65). The degree of pressure load leading to left ventricular hypertrophy seems to be better reflected by resting than by casual blood pressure.

There was no hypertensive subject with both signs of left ventricular hypertrophy on orthogonal electrocardiography and either an a/H ratio over 15 per cent or an abnormal atrial sound, indicating two different forms of cardiac involvement as the result of hypertension. Casual blood pressures became normal during rest in hypertensives with a/H ratio over 15 per cent on apex cardiography or abnormal atrial sound, but not in hypertensives with signs of left ventricular hypertrophy on orthogonal electrocardiography.

The natural history of hypertension is extremely varied. One-third of a hypertensive population survived to old age without signs of organic damage while in others the prognosis was poor (Fry, 1974). Hypertensives with signs of left ventricular hypertrophy on electrocardiography have a poorer prognosis than hypertensives with a normal electrocardiogram (Bechgaard, 1967; Sokolow and Perloff, 1961; Kannel *et al.*, 1970). In mild to moderate hypertension, antihypertensive treatment had a beneficial effect in hypertensives with signs of cardiac abnormality on electrocardiography or x-ray, while no benefit could be shown in hypertensives without such abnormality (Veterans Administration Cooperative Study Group on Antihypertensive Agents, 1972). Thus, it seems important for prognostic and therapeutic purposes to diagnose cardiac abnor-

mality in hypertensives. Conventional electrocardiography and chest x-ray are, however, normal in many hypertensives, partly because of low sensitivity, and are less suitable for quantification of left ventricular hypertrophy (Sokolow and Lyon, 1949; Gamboa, Hugenholtz, and Nadas, 1965; The National Center for Health Statistics, 1966; Tibblin, 1967, Romhilt and Estes, 1968; Sannerstedt, Bjure, and Varnauskas, 1970; McCaughan, Littman, and Pipberger, 1973).

Vectorcardiography and orthogonal electrocardiography have been shown to be superior to conventional electrocardiography in the diagnosis of left ventricular hypertrophy (Gamboa *et al.*, 1965; McCaughan *et al.*, 1973). Apex cardiography, especially the *a* wave in the apex cardiogram, and the analogous acoustical event, the atrial sound, provide non-invasive ways of assessing changes in
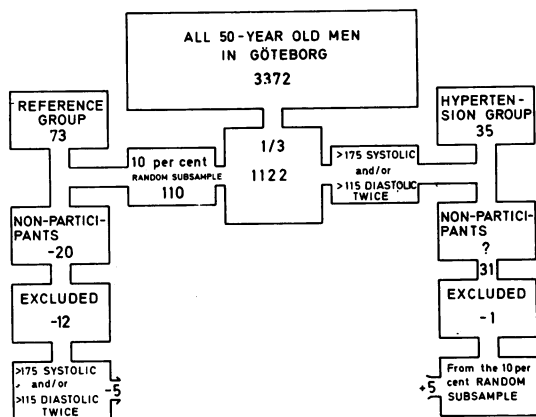
FIG. 1 *Schematic representation of the random selection of the hypertension group and the reference group from the population of men born in 1921 and living in Göteborg, Sweden.*

left ventricular distensibility (Bethell and Nixon, 1973; Gibson *et al.*, 1974; Tavel, 1974). In the present investigation signs of left ventricular hypertrophy were studied by conventional and orthogonal electrocardiography (Frank), and signs of decreased left ventricular distensibility by apex cardiography and registration of atrial sounds phonocardiographically.

The aim of the present study was to determine the prevalence of heart involvement, assessed by these non-invasive methods, in groups of untreated hypertensive and normotensive men, homogeneous with respect to age, and to relate the findings to casual blood pressure and resting blood pressure.

Subjects

From a screening examination, which was part of a multifactorial, primary preventive trial (Wilhelmsen, Tibblin, and Werkö, 1972) in a randomly selected third (n=1122) of the 50-year-old male population in Göteborg, Sweden, all subjects with untreated, essential hypertension were allocated to a hypertension group (n=35) (see Fig. 1). The diagnosis of essential hypertension was based on a casual blood pressure above 175 mmHg (23·3 kPa) systolic and/or 115 mmHg (15·3 kPa) diastolic on two separate occasions two weeks apart (the casual blood pressure values given in the tables refer to the first measurement), and a negative standard diagnostic examination for secondary hypertension (Wilhelmsen, Berglund, and Werkö, 1973). Thus, one subject with chronic glomerulonephritis was excluded

(Fig. 1). For the hypertension group, 18 subjects were classified as belonging to WHO stage 1, 13 to stage 2, and 4 to stage 3.

A reference group (n=73) was obtained from the same trial by drawing a 10 per cent subsample at random. The non-participation rate in the subsample was 18 per cent (20/110). Five subjects in the reference group fulfilled the criteria for hypertension and joined the hypertension group. Twelve subjects in the reference group were excluded, 1 with mitral stenosis, 1 with aortic regurgitation, 5 who were on antihypertensive treatment, and 5 with casual blood pressure above 175 mmHg (23·3 kPa) systolic and/or 115 mmHg (15·3 kPa) diastolic at screening but not at the subsequent blood pressure control two weeks later. No subject in the hypertension group had a history of valvular heart disease.

In addition to the above-mentioned groups, 3 untreated, male hypertensives (aged 45, 50, and 50 years) with casual blood pressures above 240 mmHg (31·9 kPa) systolic and 160 mmHg (21·3 kPa) diastolic, recruited from the same screening examination, were examined in order to obtain data from subjects with extremely high blood pressures. These 3 subjects all belonged to WHO stage 3.

Orthogonal electrocardiograms and conventional electrocardiograms were recorded in all subjects (n=111). Registration of apex cardiogram, atrial sounds, and resting blood pressure were performed in a randomly selected half of both the reference (n=36) and the hypertension group (n=19) and in the 3 hypertensives with extremely high blood pressure. Analysis of each variable was performed without knowledge of the results of the other examinations or to which group the subject belonged. Resting blood pressure values were, for technical reasons, lacking for 4 subjects selected for apex cardiography; all 4 belonged to the reference group. At analysis of signs of left ventricular hypertrophy on electrocardiography, 2 subjects in the hypertension group and 1 in the reference group with left or right bundle-branch block or left anterior hemiblock, were excluded, since changes in left ventricular activation have an influence on the amplitudes (Fernandez, Scabat, and Lenegre, 1970). In 4 subjects in the reference group (11%) and 1 hypertensive subject (5%), an acceptable apex cardiogram could not be recorded.

Methods

Conventional electrocardiograms, apex cardiograms, phonocardiograms, and resting blood pressures were all registered on a direct writing ink-jet 7-channel

mingograph (EM 81, Siemens-Elema, Sweden) with and linear frequency response from 0 to 500 Hz 30 per cent amplitude reduction at 650 Hz. The mingograph was supplied with a phonopreamplifier (EMT 22) with electrical filters that together with a piezoelectric microphone (EMT 25 C) give five frequency ranges with the following nominal frequencies: 25, 50, 100, 200, 400 Hz and one aural frequency range. A crystal transducer (EMT 510 C) with low frequency time constant between 1·9 and 4·6 s (depending on a capacitance—resistance product, decided by the individual amplification used for each curve) connected by a 35 to 40 cm long rubber tube to a specially designed capillary damped funnel pick-up, 2·5 cm in diameter, giving a frequency response of 0·08 (at time constant 1·9 s) to 65 Hz (–3db), was used. Paper speed was 50 mm/s for electrocardiogram and for the other tracings 100 mm/s.

Blood pressure was measured with a 12 cm broad and 26 cm long rubber cuff. Cuff inflation was rapid, cuff deflation approximately 3 beats per 10 mmHg (1·3 kPa). Diastolic pressure was registered as phase 5, i.e. when the sounds disappeared. Casual blood pressure was measured in the seated position with a mercury manometer and stethoscope at the screening examination. Resting blood pressure was measured after one hour's rest in the supine position in a sound-protected room with an automatic device for cuff inflation and deflation (Boucke-Brecht), with a microphone (EMT 25 C) placed over the right brachial artery and with simultaneous registration of cuff pressure, Korotkoff sounds, and electrocardiogram. The largest circumference of the relaxed right overarm was measured in those subjects in whom resting blood pressure was measured.

Casual heart rate was measured as the mean of five consecutive beats from an electrocardiogram taken at the screening examination. Resting heart rate was measured from the resting blood pressure recordings. The resting blood pressure was measured twice with an interval of one minute. The following correlations between these resting blood pressures were found: reference group systolic $r=0·95$, diastolic $r=0·94$; hypertension group systolic $r=0·94$, diastolic $r=0·96$. No significant correlation for the relation between the circumference of the right upper arm and resting blood pressure was found (reference group $r=0·11$ systolic, $r=0·17$ diastolic; hypertension group $r=0·09$ systolic, $r=0·31$ diastolic). There was no significant difference in the mean of the right upper arm circumference between the hypertension group ($\bar{x}$ 31·8, s_x 2·5, range 27·0 to 35·5 cm) and the reference group ($\bar{x}$ 30·8, s_x 2·1, range 26·0 to 35·5 cm).

The three scalar electrocardiographic leads X, Y, and Z were simultaneously recorded on magnetic tape (Hellige) using the Frank's corrected orthogonal lead system (Frank, 1956). Computer analysis was performed according to Arvedson (1968, 1973) with a sampling frequency of 200 per second. The following criteria for left ventricular hypertrophy were used: the amplitude of the R wave in lead X (R_x) above 1·8 mV, the R wave in lead X and the S wave in lead Y ($R_x + S_y$) above 1·9 mV or the R wave in lead Z (R_z) above 1·3mV. Since most electrocardiographic measurements are not normally distributed the amplitude limits were drawn at the upper 97·5th centile in the reference group in analogy with a previous study (McCaughan et al., 1973). The upper 97·5th centile in the reference group was also studied for $R_x + R_z$ (2·8 mV), the spatial maximal amplitude (2·2 mV), ventricular activation time (0·06 s for both R_x and the spatial maximal amplitude), the duration of the P wave (0·13 s) and the following amplitudes of the P wave in the orthogonal electrocardiogram: sum of positive P amplitudes in lead X and lead Z (0·17 mV), positive P amplitude in lead Z (0·07 mV) (Ishikawa, Kini, and Pipberger, 1973). Conventional electrocardiograms were recorded as standard 12 lead electrocardiograms using the right arm as the indifferent electrode for the praecordial leads. Electrocardiograms were coded in accordance with the Minnesota code (Blackburn et al., 1960). Amplitude measurements in praecordial leads were done in accordance with the revision for CR leads (Åstrand et al., 1967). As signs of left ventricular hypertrophy, a combination of amplitude criteria, 3:1 or 3:3, and ST or T criteria, 4:1–3 or 5:1–3, were used (Sokolow and Lyon, 1949).

Apex cardiograms and phonocardiograms were recorded during the resting period preceding the measurement of resting blood pressure. The apex cardiogram was recorded simultaneously with electrocardiogram lead II and a phonocardiogram from the third left intercostal space parasternally with the subject in the left lateral position during relaxed expiratory apnoea. The pick-up was held by hand at the point of maximal impulse of the apex beat. The *a* wave percentage amplitude of the total deflection of the apex cardiogram (H), was calculated as the mean of five consecutive beat- (Fig. 2). An *a*/H ratio above 15 per cent was considered abnormal (Tavel et al., 1965; Epstein et al., 1968; Voigt and Friesinger, 1970). Registration of the apex cardiogram by two different examiners with one hour's interval, the second examiner not being allowed to see the recording made by the first examiner, gave a correlation coefficient of 0·88 (n=81) between the two calculated *a*/H
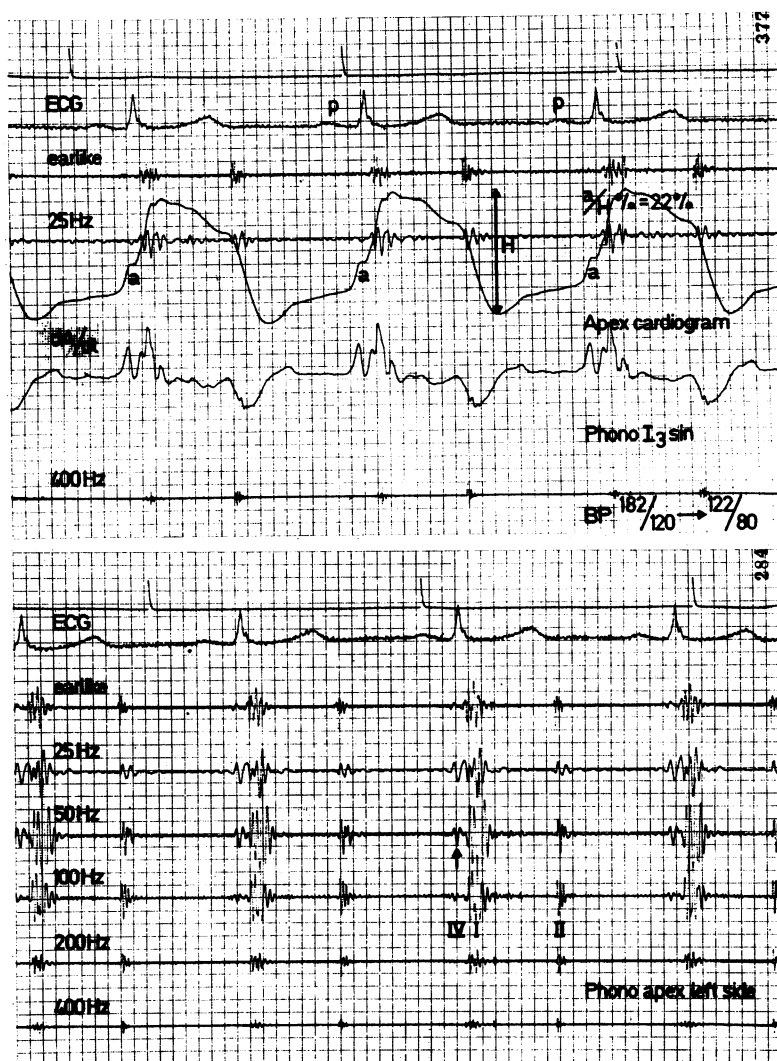
FIG. 2   *Apex cardiogram and phonocardiogram in a hypertensive subject in the left lateral position. Upper panel: apex cardiogram with an abnormal a wave (a). The a wave percentage amplitude of the total deflection of the apex cardiogram (H) was 22 per cent calculated as the mean a/H ratio of 5 consecutive beats. The apex cardiogram was recorded simultaneously with electrocardiogram lead II, phonocardiogram from the third intercostal space parasternally, and the first derivative of the apex cardiogram (dA/dt). Screening blood pressure (BP) was 182/120 mmHg (24·2/16·0 kPa), resting blood pressure was 122/80 mmHg (16·2/10·6 kPa). Lower panel: phonocardiogram from the apex region showing an atrial sound of intensity 4. The phonocardiogram was recorded with six frequency ranges giving five nominal frequencies from 25 to 400 Hz and one aural frequency range.*

ratios, with a standard deviation of a single determination of 2·5 per cent.

Phonocardiograms were recorded during relaxed expiratory apnoea simultaneously with electrocardiographic lead II from the routine clinical auscultation points including registration over the apex in the left lateral position (Fig. 2). One of the authors (I.W.) with long experience of phonocardiography, classified the atrial sound in the phonocardiogram with regard to its intensity, on a five-point subjective scale, where 1 was judged as 'very weak' and 5 as 'very strong'. Atrial sounds of intensity 4 and 5 were arbitrarily classified as abnormal. A blind re-evaluation of the phonocardiograms with respect to the intensity of the atrial sound was performed after one year. One atrial sound in the hypertension group that had previously been evaluated as 4 was now judged to be 3. This subject had an abnormal a wave in the apex cardiogram. At the latter evaluation the proportion of subjects with abnormal atrial sounds was not significantly higher in the hypertension group than in the reference group.

An abnormal atrial sound is said to have the same haemodynamic significance as an abnormal *a* wave in the apex cardiogram (Tavel, 1974; Bethell and Nixon, 1973). Good correlation has been shown between the relative magnitude of the *a* wave and an index of the left ventricular distensibility (Gibson *et al.*, 1974). Therefore, an abnormal *a* wave in the apex cardiogram and an abnormal atrial sound were both regarded as signs of decreased left ventricular distensibility.

Standard methods were used for calculation of the mean ($\bar{x}$), the standard deviation (s_x), the standard error of the mean ($s_{\bar{x}}$), and the linear correlation coefficient (r). The hypothesis of no differences in means was tested with Student's t-test or the Wilcoxon Rank Sum Test for two samples. The hypothesis of no differences between paired observations in the same subjects was tested with Student's t-test for paired observations. Where the standard deviation and range are given the t-test was used; where the standard error of the mean and range are given the Wilcoxon test was used. The hypothesis of no differences in proportions between two groups was tested with the Fisher exact test for $n < 60$ and with the χ^2 test for $n \geq 60$. The hypothesis of no differences in proportions when examining one group of subjects with two separate methods was tested with McNemar's test for correlated proportions (Remington and Schork, 1970).

The formula $\sqrt{\dfrac{\Sigma d_i^2}{2n}}$ was used to calculate the standard deviation of a single determination in a series of determinations of which the mean difference was not significant (Dahlberg, 1940). Only two-tailed tests were used and differences were considered significant for P values 0·05 or less.

Results

Blood pressure and heart rate

In both the hypertension and the reference group the resting systolic and diastolic blood pressures and heart rates were significantly lower (P < 0·001) than the casual (Table 1). The hypertensives showed significantly higher (P < 0·05) casual heart rate than the reference group, but after one hour's rest in a sound-protected room there was no significant difference. The differences in resting systolic and diastolic blood pressure between the two groups were significant (P < 0·001).

TABLE 1 *Casual and resting blood pressure and heart rate in subjects randomly selected for apex cardiography*

			Hypertension group No.=19			Reference group No.=32		
			$\bar{x}$	Range	s_x	$\bar{x}$	Range	s_x
Systolic	Casual	mmHg	197	(220–164)	15	142	(174–116)	15
	Resting		154	(192–111)	23	123	(152–104)	13
Diastolic	Casual	mmHg	119	(138–98)	9	92	(110–70)	8
	Resting		96	(120–64)	15	77	(96–51)	10
Heart rate	Casual	beats/min	84	(114–61)	17	75	(114–54)	15
	Resting		61	(74–43)	8	60	(83–46)	8

Conversion factor from Traditional to SI Units: 1 mmHg ≈ 0·133 kPa.

TABLE 2 *Selected scalar measurements on orthogonal and conventional electrocardiography that discriminate between hypertension and reference groups*

	Hypertension group		Reference group	
	No.	%	No.	%
Orthogonal electrocardiogram				
$R_x > 1 \cdot 8$ or $R_z > 1 \cdot 3$ or				
$R_x + S_y > 1 \cdot 9$ mV	11/33	33	2/72	3
Conventional electrocardiogram				
3:1 or 3:3 plus				
4:1–3 or 5:1–3	3/33	9	2/72	3
3:1 or 3:3 alone	6/33	18	5/72	7

Orthogonal electrocardiogram and standard 12-lead electrocardiogram

The proportion of hypertensives with signs of left ventricular hypertrophy on orthogonal electrocardiogram (11/33, 33%) was significantly higher (P < 0·001) than that in the reference group (2/72, 3%), see Table 2. The proportion of hypertensives with signs of left ventricular hypertrophy with conventional electrocardiogram was 9 per cent (3/33), compared with 3 per cent (2/72) in the reference group. Minnesota code 3:1 or 3:3 alone was seen in 18 per cent (6/33) of the hypertensives and in 7 per cent (5/72) of the reference group. The proportion of hypertensives with signs of left ventricular hypertrophy by orthogonal electrocardiogram was significantly higher (McNemar, P < 0·025) than by conventional electrocardiogram. In the reference group there was no overlapping between the two methods, i.e. two subjects showed signs of left ventricular hypertrophy by orthogonal electrocardiogram, and another two by conventional electrocardiogram. Of the hypertensives, one fulfilled the criteria for left ventricular hypertrophy with conventional electrocardiogram only, two with both methods, and nine with orthogonal electrocardiogram only. Of these nine subjects, none had a Minnesota code 4:1–3 or 5:1–3, i.e. ST or T changes on conventional electrocardiogram, and thus the only sign of left ventricular hypertrophy in these nine hypertensives was high QRS amplitude on orthogonal electrocardiogram. Other measurements, such as $R_x + R_z$, the spatial maximal amplitude, ventricular activation time, or the duration or amplitude of the P wave did not improve the discrimination between the hypertension and reference group.

Apex cardiogram and atrial sound

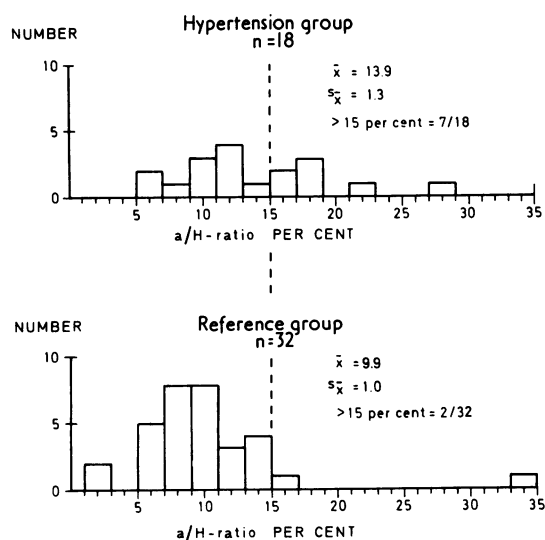The proportion of hypertensives with a/H ratio above 15 per cent (7/18, 39%) was significantly

FIG. 3 *Distribution of the a/H ratio from the apex cardiogram and the proportion of subjects with a/H ratio above 15 per cent, the mean ($\bar{x}$) and the standard error of the mean ($s_{\bar{x}}$) in the hypertension and reference group. For practical reasons, one subject in the reference group with a/H ratio $= 0$ per cent was placed in the interval 1 to 3 per cent in the figure.*

higher (P < 0·05) than found in the reference group (2/32, 6%), see Fig 3. The mean a/H ratio in the hypertension group ($\bar{x} = 13 \cdot 9$, $s_{\bar{x}} = 1 \cdot 3$, range 6 to 28) was significantly higher (P < 0·01) than the mean in the reference group ($\bar{x} = 9 \cdot 9\%$, $s_{\bar{x}} = 1 \cdot 0$, range 0 to 33).

Atrial sounds of intensity 1 to 3 were common in both groups (Fig. 4). The proportion of hypertensives with atrial sounds of intensity 4 and 5 (5/19, 26%) was significantly higher (P ⩽ 0·05) than in the reference group (1/36, 3%). All abnormal atrial sounds had their maximum intensity over the apex in the left lateral position. No measurements of the atrial sound percentage amplitude of the first heart sound could discriminate between the groups. Three of the seven hypertensives who had a a/H ratio above 15 per cent also had abnormal atrial sounds. One of the two subjects in the reference group, who had an abnormal a wave on the apex cardiogram, had diabetes mellitus and a high casual blood pressure (172/98 mmHg (22·9/13·0 kPa)). The other subject with a/H ratio 33 per cent had angina pectoris. He also had an abnormal atrial sound (intensity 5) and ST and T changes on conventional electrocardiography.
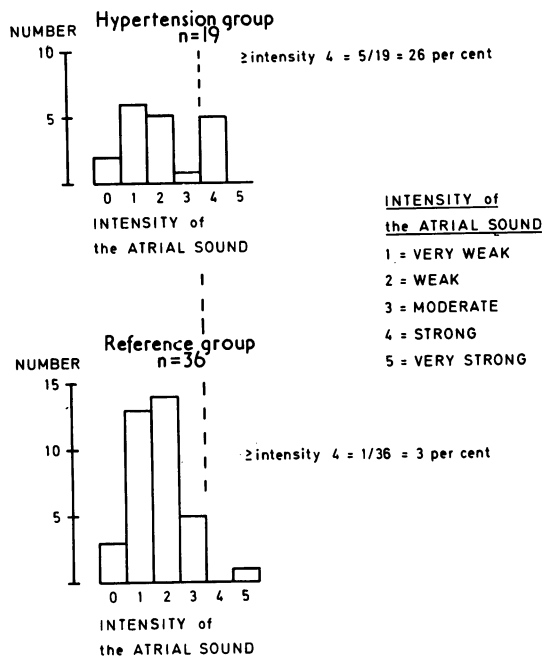
FIG. 4 *Distribution of atrial sounds of intensity 1 to 5 and the proportion of subjects with atrial sounds of intensity 4 and 5 in the hypertension and reference groups.*

Signs of left ventricular hypertrophy and a/H ratio

The mean a/H ratio in the group of hypertensives with signs of left ventricular hypertrophy on orthogonal electrocardiogram ($\bar{x}=9\cdot0\%$, $s_{\bar{x}}=1\cdot0$, range 6 to 12, $n=5$) was significantly *lower* ($P \leqslant 0\cdot01$) than in the group of hypertensives without signs of left ventricular hypertrophy ($\bar{x}=16\cdot1\%$, $s_{\bar{x}}=1\cdot8$,

range 7 to 28, $n=11$). In this analysis two subjects with ventricular conduction defects were excluded.

Blood pressure and type of heart involvement

There was no hypertensive showing both signs of left ventricular hypertrophy on orthogonal electro-cardiogram and an abnormal *a* wave on the apex cardiogram or an abnormal atrial sound, i.e. signs of decreased distensibility of the left ventricle. Table 3 shows that hypertensives with signs of left ventricular hypertrophy on orthogonal electro-cardiography had significantly higher ($P \leqslant 0\cdot02$) casual diastolic blood pressure and significantly higher systolic ($P < 0\cdot05$) as well as diastolic ($P \leqslant 0\cdot01$) resting blood pressures than the group of hypertensives with signs of decreased left ventricular distensibility. There was no significant difference in heart rate between the two groups. If the two hypertensives with only abnormal atrial sound are removed from the group with a/H ratio greater than 15 per cent, there still was a significant difference ($P \leqslant 0\cdot02$) in resting diastolic blood pressure between the two groups.

Findings in three hypertensives with extremely high blood pressure

The casual blood pressures for these three subjects were 240, 268, and 250 mmHg (31·9, 35·6, and 33·3 kPa) systolic and 172, 164, and 160 mmHg (22·9, 21·8, and 21·3 kPa) diastolic, and the casual heart rates were 99, 72, and 89 beats/min. The third subject was treated immediately; the resting blood pressures for the other two were 196/130 mmHg (26·1/17·3 kPa) and 198/107 mmHg (26·3/14·2 kPa), respectively. All three had signs of left ventricular hypertrophy on orthogonal electrocardiogram ($R_x = 2\cdot3$, $R_x + S_y = 1\cdot9$, and $R_z = 2\cdot5$ mV, respectively) and showed abnormal a/H ratios (50, 32, and 27%).

TABLE 3 *Blood pressure and heart rate in two groups of hypertensives with signs of left ventricular hypertrophy ($R_x > 1\cdot8$ or $R_z > 1\cdot3$ or $R_x + S_y > 1\cdot9$ mV) and signs of lowered left ventricular distensibility (a/H ratio $> 15\%$ or atrial sound of intensity 4 or 5)*

| | | | Type of left ventricular involvement | | | | | |
| | | | Hypertrophy No.$=5/19$ | | | Lowered distensibility* No.$=8/19$ | | |
			$\bar{x}$	Range	$s_{\bar{x}}$	$\bar{x}$	Range	$s_{\bar{x}}$
Casual	Systolic		206	(216–198)	3	193	(220–164)	7
	Diastolic	(mmHg)	125	(132–118)	2	116	(122–104)	2
	Heart rate	(beats/min)	79	(114–61)	9	87	(108–61)	6
Resting	Systolic		171	(192–146)	7	137	(172–111)	8
	Diastolic	mmHg	107	(120–98)	4	86	(107–64)	5
	Heart rate	(beats/min)	59	(74–50)	4	60	(73–43)	3

*One subject with left bundle-branch block was excluded since left ventricular hypertrophy on orthogonal electrocardiography could not be analysed.
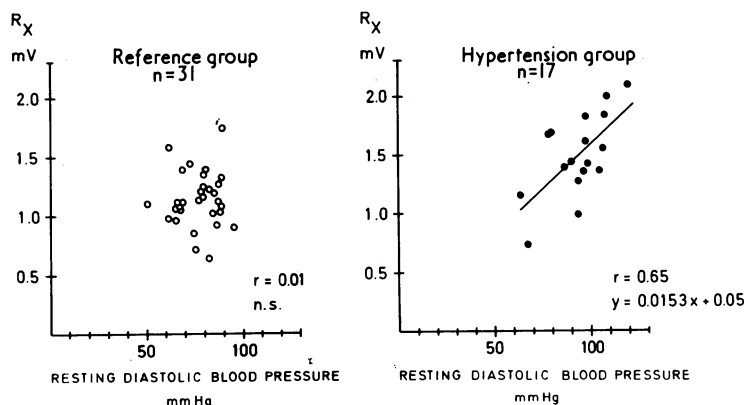*Conversion from Traditional to SI Units:* 1 mmHg$\approx 0\cdot133$ kPa.

FIG. 5 *Relation between resting diastolic blood pressure and the amplitude of the R wave in lead X on orthogonal electrocardiogram. (Conversion from Traditional to SI Units: 1mmHg ≈ 0·133 kPa.)*

Two had abnormal atrial sounds as well. These three hypertensives with extremely high blood pressures thus showed signs of both left ventricular hypertrophy and decreased left ventricular distensibility.

Relations between signs of left ventricular hypertrophy on orthogonal electrocardiogram, a/H ratio, and blood pressure

Linear correlation analysis for the relation between R_x on orthogonal electrocardiogram and resting diastolic blood pressure in the hypertension group (see Fig. 5) gave r=0·65 (n=17, P<0·01). When the subjects with extremely high blood pressure were included in the linear regression analysis, the r value rose to 0·74. A significant positive correlation was also found in the hypertension group for the relation between R_x and resting systolic blood pressure (r=0·50, n=17, P<0·05), as well as casual diastolic blood pressure (r=0·40, n=33, P<0·05). There was also a significant positive correlation in the hypertension group between resting diastolic blood pressure and $R_x + S_y$ (r=0·67, n=17, P<0·01) and spatial maximal amplitude (r=0·55, P<0·05). There was no significant linear correlation in the hypertension group either for the relation between casual systolic blood pressure and R_x (r=0·03, n=33), $R_x + S_y$ (r=0·01), R_z (r=0·33), or spatial maximal amplitude (r=0·05). In the hypertension group R_x above 1·8 mV was only seen when the casual diastolic pressure exceeded 110 mmHg (14·6kPa) or resting diastolic pressure exceeded 95 mmHg (12·6 kPa). In the reference group there was no significant correlation of R_x either with casual or with resting blood

pressure. No significant linear correlations of a/H ratio to blood pressure were found either in the hypertension, or in the reference group.

Discussion

The subjects of the study were selected at random from a total male population. As most of the variables studied are age and sex dependent, we chose to study normotensive and hypertensive men of the same age. To our knowledge, no previous study of cardiac function in relation to blood pressure has used these quantitative, non-invasive methods in subjects derived from a screening examination for blood pressure in a total population.

In the hypertension group, orthogonal electrocardiography showed a higher prevalence of signs of left ventricular hypertrophy than conventional electrocardiography. Since the prevalence of signs of left ventricular hypertrophy in the reference group was the same with both methods, orthogonal electrocardiography seems to be a better method for discrimination of left ventricular hypertrophy in hypertension. Furthermore, orthogonal electrocardiography is more practical, since simple QRS amplitude measurements are sufficient, while on conventional electrocardiography, QRS amplitude measurements have to be combined with other criteria, such as ventricular activation time or ST or T changes for the diagnosis of left ventricular hypertrophy (Sokolow and Lyon, 1949). Measurement of the spatial maximal amplitude was found redundant, as had also been found in a previous study (McCaughan et al., 1973).

Atrial sounds can be recorded in the majority of

normal individuals aged 50 (Rectra *et al.*, 1972) and this is also evident from the present study. This means that the atrial sound as well as the *a* wave in the apex cardiogram has to be in some way quantified to be of use in differentiating normal from abnormal. The judgement had to depend on the empirical judgement of one of the authors. Even if the hypertensives with only abnormal atrial sound are removed from the hypertensives with *a*/H ratio greater than 15 per cent the figures still allow the separation of two types of heart involvement in hypertension. Apex cardiogram and recording of atrial sounds complement each other, since the overlapping between the two methods was less than often claimed (Tavel *et al.*, 1965; Epstein *et al.*, 1968; Tavel, 1974). Different filter properties of the thoracic wall for sound and volume displacement as well as the difference in origin mechanisms for the *a* wave and the atrial sound may explain the finding that there was no total overlapping.

The morphological evidence of left ventricular hypertrophy is increased left ventricular mass (Romhilt and Estes, 1968). Mass itself has been shown to be a poor predictor of distensibility, while wall thickness was an excellent predictor (Grossman *et al.*, 1974). The mean *a*/H ratio in patients with signs of left ventricular hypertrophy on conventional electrocardiography has been shown to be significantly higher than in patients without left ventricular hypertrophy (Gibson *et al.*, 1974). In the present study mean *a*/H ratio was significantly lower in hypertensives with signs of left ventricular hypertrophy on orthogonal electrocardiography than in hypertensives without these signs, which is in contrast to the above-mentioned studies. Hypertensives in the present study with signs of left ventricular hypertrophy had no signs of decreased left ventricular distensibility. One reason for this might be that the hypertrophy was not of the degree that gives a sufficient increase in wall thickness to give distensibility changes. The degree of hypertrophy in our hypertensives was probably mild compared with the hypertrophy in the above-mentioned studies. Another explanation could be that a relative strengthening of the systolic wave in relation to the *a* wave resulting from the hypertrophy could have led to a decrease in the *a*/H ratio. In two of the three subjects with extreme levels of casual blood pressure the decreased left ventricular distensibility might have been caused by left ventricular hypertrophy, namely in Cases 1 and 3 where the QRS amplitudes on orthogonal electrocardiography were very large.

The signs of decreased left ventricular distensibility in the hypertension group could not be related to hypertrophy, at least not electrocardiographically

proven. Decreased distensibility of the left ventricle is not a specific finding of left ventricular hypertrophy but is also found in ischaemic heart disease (Voigt and Friesinger, 1970; Martin, Shaver, and Leonard, 1972). There was, however, no subject in the hypertension group with symptoms of angina pectoris as judged by one of the authors (G.B.). In any consideration of ventricular distensibility, it must be borne in mind that the diastolic pressure-volume curve is not linear. Thus, a structurally normal ventricle may exhibit the same low distensibility as a scarred or hypertrophied ventricle if its filling pressure is raised sufficiently (Levine, 1972). Another explanation for the signs of decreased left ventricular distensibility could thus be abnormal distension of the left ventricle during the atrial contraction partly caused by increased central blood volume. Increased central blood volume has been described in essential hypertension (Ulrych *et al.*, 1969; Ellis and Julius, 1973).

A model of the circulation has been described in which hypertension initially resulting from increased blood volume and/or cardiac output can subsequently become converted to a hypertension with high peripheral resistance and normal or lowered cardiac output (Guyton and Coleman, 1969). In the present study the resting blood pressure was returned to normal in the hypertensives with signs of decreased left ventricular distensibility, but not in the hypertensives with signs of left ventricular hypertrophy. This might suggest that the former group is in an earlier state of hypertensive disease while the other is in a later state with raised peripheral resistance. Whether the former group will pass over into a hypertension of the latter type or not must be studied prospectively. In advanced left ventricular hypertrophy an abnormal *a* wave in the apex cardiogram is a very common finding (Tavel *et al.*, 1965; Epstein *et al.*, 1968). It might be that an initial abnormal *a* wave caused by volume stiffness of the left ventricle can return to normal, and then when the left ventricular hypertrophy becomes pronounced or fibrosis is added, structural changes in the left ventricular wall can again give rise to an abnormal *a* wave.

The correlation coefficient between blood pressure and amplitude measurements on orthogonal electrocardiography were higher in the present study than in a previous one (McCaughan *et al.*, 1973) ($r = 0.65$ for R_x in the present study, compared with $r = 0.23$ for R_z in the previous investigation). In the latter investigation, however, a group of patients with severe hypertensive heart disease had large R_z amplitudes and a concomitant decrease in R_x. The homogeneity of our hypertension group of subjects with early hypertension probably implies

that the amplitude of the R_x will reflect the degree of left ventricular hypertrophy better than will R_x in the latter investigation.

In the present investigation there was better correlation between QRS amplitude measurements and resting blood pressure than to casual blood pressure, indicating that resting blood pressure better reflects the degree of pressure load leading to left ventricular hypertrophy. The fact that these hypertensives were untreated and never had been treated probably also implies that the blood pressures were more representative of the pressure load on the left ventricle than blood pressure in treated patients can be.

It is evident from the present study that the orthogonal electrocardiogram, the apex cardiogram, and the recording of atrial sounds complement each other in the investigation of hypertensive heart disease. The combination of the three methods made it possible to detect heart involvement in the majority of hypertensive subjects. The numbers with full studies are, however, small. Prospective follow-up must be undertaken in larger groups to study the haemodynamic pattern and prognosis in hypertensives with the different abnormal findings. These studies are now proceeding.

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